MEDICAL PROCEEDINGS
MEDIESE BYDRAES

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EDITORIAL · REDAKSIONEEL

IMMUNITY FOLLOWING ANTI-POLIOMYELITIS VACCINATION

In tests carried out on a number of children, Salk[1] studied the important question of how many vaccinations are required to ensure an effective and lasting immunity with a vaccine prepared from a killed virus. The criterion used for determining immunity was the titre of antibody found in the blood at various intervals up to 4 years after vaccination.

Following anti-poliomyelitis vaccination the antibody titre rises rapidly, reaching its maximum when 2 booster injections have been given. Thereafter, it drops to a certain level which remains constant for some considerable time. To ensure adequate immunity with 3 injections, a sufficient quantity of antigen must be administered. This also determines the strength of the response to subsequent booster injections.

According to the data available at present there is no need for a fourth injection of poliomyelitis vaccine. On the other hand, there is no reason to fear that 4, 5 or even 6 vaccinations may be harmful. Of importance is Salk's observation that *multiple injections of a vaccine of low potency are not as reliable a way to induce immunity as are fewer injections of a vaccine of adequate potency.*

This is also borne out by the investigations of Buser *et al.*,[2] who studied the question of how the neutralizing antibodies directed against

1. Salk, J. E. (1958): J. Amer. Med. Assoc., **167**, 1.
2. Buser, F., Martin Du Pan, R., and Mégevand, A. (1958): Schweiz. Med. Wschr., **88**, 530.

ONVATBAARHEID VOLGENDE OP ANTI-POLIOMIËLITIS-INENTING

In toetse uitgevoer met 'n aantal kinders het Salk[1] 'n studie van 'n belangrike vraag gemaak, nl. hoeveel inentings nodig is om doeltreffende en blywende onvatbaarheid te verseker met 'n entstof wat van vernietigde virus voorberei is. Die maatstaf wat vir die vasstelling van onvatbaarheid gebruik is, was die titer van teenstof wat by verskillende tussenpose tot 4 jaar ná inenting in die bloed gevind is.

Volgens op anti-poliomiëlitis-inenting styg die teenstof-titer vinnig en bereik sy maksimum nadat 2 aanjaaginspuitings toegedien is. Daarna daal dit tot 'n sekere peil wat 'n aansienlike tyd lank standhoudend bly. Om onvatbaarheid met 3 inspuitings te verseker, is dit nodig om 'n voldoende hoeveelheid van die antigeen toe te dien. Dit bepaal ook die sterkte van die reaksie op latere aanjaaginspuitings.

Volgens die gegewens wat op die oomblik beskikbaar is, is 'n vierde inspuiting van poliomiëlitis-entstof nie nodig nie. Aan die ander kant is daar geen rede om te vrees dat 4, 5 of selfs 6 inentings skadelik sal wees nie. Van belang is Salk se waarneming dat *veelvoudige inspuitings van 'n entstof van lae sterkte nie so 'n betroubare manier is om onvatbaarheid teweeg te bring as 'n kleiner aantal inspuitings van 'n entstof van doelmatige sterkte nie.*

1. Salk, J. E. (1958): J. Amer. Med. Assoc., **167**, 1.
2. Buser, F., Martin Du Pan, R. en Mégevand, A. (1958): Schweiz. Med. Wschr., **88**, 530.

the polio virus behave in infants and small children following inoculation with Salk vaccine. They found that after 2 vaccinations the response was weak, insofar as a rise in antibody titre was observed only in isolated cases. Following the third vaccination the titre of the antibodies directed against type II virus was somewhat better, while the titre of the antibodies directed against types I and III only seldom reached optimum values.

The results thus deviate from those of previous investigations. This may be due to the fact that the Salk vaccine has recently been submitted to more stringent analytical tests in the United States, in particular to filtration and increased inactivation. This modification in the manufacturing method has apparently had an unfavourable effect on the immunizing effectiveness of the vaccine.

The essential production problem remains one of making an effectively antigenic vaccine which is yet safe. Immunization programmes should therefore only be undertaken with safe vaccines which are not alone antigenic, but adequately so.

Dit word ook bevestig deur die navorsingswerk van Buser *et al.*,[2] wat ondersoek ingestel het na die vraag hoe die neutraliserende teenstowwe wat op die poliovirus inwerk hulle gedra in babetjies en klein kinders, volgende op inenting met Salk-entstof. Hulle het bevind dat die reaksie ná 2 inspuitings swak was, in soverre dat 'n styging van die teenstoftiter slegs in enkele gevalle waargeneem is. Volgende op die derde inenting was die titer van die teenstowwe wat op die Tipe II-virus inwerk, effens beter, terwyl die titer van die teenstowwe wat op Tipes I en III inwerk, slegs in uitsonderlike gevalle die optimum-waardes bereik het.

Die resultate wyk dus van vroeëre ondersoekingswerk af. Dit moet miskien toegeskryf word aan die feit dat die Salk-entstof onlangs aan strenger analitiese toetse in die Verenigde State onderwerp is—in besonder aan filtering en verhoogde inaktivering. Hierdie wysiging van die vervaardigingsmetode het skynbaar 'n ongunstige uitwerking op die immunisasiedoeltreffendheid van die entstof gehad.

Die essensiële voorbereidingsprobleem bly, soos altyd, die vervaardiging van 'n doeltreffende antigeenentstof wat veilig is. Immunisasieprogramme behoort derhalwe onderneem te word alleen met veilige entstof wat nie alleen antigenies is nie, maar doeltreffend antigenies.

EXTRA-LOBAR SEQUESTRATION OF THE LUNG

REPORT OF A CASE

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Only a small number of cases of extra-lobar sequestration of the lung have been reported. The following appears to be the first such case to be reported from South Africa. It shows some unusual pathological features.

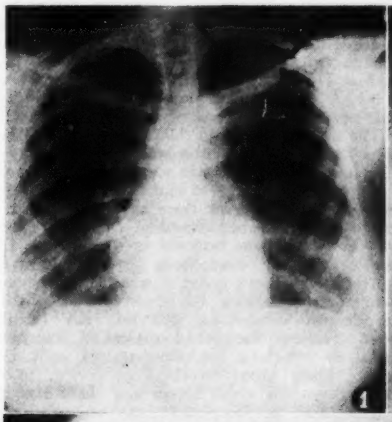
CASE REPORT

Miss E. W., a 19-year-old European nurse, was admitted to the Thoracic Surgical Unit for investigation of a mass in the left upper chest, found on routine X-ray examination (Figs. 1 and 2). Her only symptoms were that for 3 months before admission she had had a dry cough, and a slight pain in the left subscapular region which was related to exercise.

Examination of the chest did not reveal any abnormalities. X-ray showed a cystic mass situated in the region of the posterior segment of the left upper lobe. It was not possible to determine whether the mass was intrapulmonary, or whether it was extrapulmonary in the left upper para-vertebral gutter. Tomography of this area confirmed a well-defined oval cystic mass (Fig. 3).

A blood count done by Dr. E. McCrone, South African Institute for Medical Research, was as follows:

Haemoglobin: 16 g.%.
Leucocytes: 9,800 per c. mm.



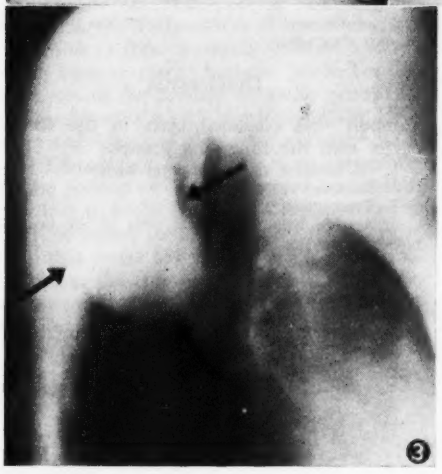


Fig. 1. Postero-anterior view of chest showing an opacity in the left upper lobe.

Fig. 2. Apical postero-anterior view of chest showing the cystic opacity.

Fig. 3. Left lateral tomograph showing the opacity in the postero-superior aspect of the chest.

Neutrophil segmented cells: 59.0%.
Monocytes: 4.0%.
Lymphocytes: 32.0%.
Eosinophil segmented cells: 5.0%.

The red cells and platelets were normal in appearance.

There was a slight absolute eosinophilia.

In view of the slight absolute eosinophilia and the cystic nature of the lesion, Casoni and hydatid complement fixation tests were done twice. The Casoni test was negative but on both occasions the hydatid complement fixation test was positive. A presumptive diagnosis of pulmonary hydatid cyst was made, and it was decided to do a thoracotomy.

Cortisone was given pre-operatively as a precautionary measure against anaphylactic reactions in case the cyst should be ruptured during the operation. A thoracotomy through the left chest, with resection of the fourth rib, was done. After opening the chest it was noted that the posterior segment of the left upper lobe was either replaced or compressed by an oval cystic mass, measuring about 9 x 6 cm. The cyst was capped at one end by tissue which resembled atelectatic lung (Fig. 4).

The lung was deflated and the cyst was found to be lying in the postero-medial aspect of the chest, opposite the vertebral end of the fourth rib and overlying the aorta and the left pulmonary artery. Three large veins which drained into the superior intercostal vein ramified over the cyst. No obvious arterial supply could be seen, but minute arterial vessels were present in the cyst wall. The base of the cyst extended down between the aorta and the left pulmonary artery away from the left main bronchus. After safeguarding the vagus and the recurrent laryngeal nerves, a few very small

arteries, apparently arising from intercostal vessels, were secured. The cyst, with the overlying lung tissue, was removed *in toto*.

Before the chest was closed, the lung was re-inflated and it was noted that the wedge-shaped defect in the area of the posterior segment of the left upper lobe was still present, but smaller in size.

Convalescence was uneventful and the patient was discharged after 10 days. A bronchogram done one month later revealed the normal bronchial divisions in a left upper lobe.

PATHOLOGICAL EXAMINATION

The specimen consisted of a tense, egg-shaped cyst about 9 x 6 x 5 cm. in size, with a cap of non-aerated lung tissue, about 4 cm. in diameter, and 1 cm. thick at the centre, adherent to one end (Fig. 4). On incision it was noted that the cyst was divided into 2 spaces by a thin membrane. The larger space contained a greyish-white homogeneous gelatinous substance and a similar pale-yellow, firmer substance was present in the other space. The wall of the cyst was about 1 mm. thick, and smooth on both surfaces. No obvious bronchial attachment was observed, and a network of blood vessels coursed over the surface of the cyst. The atelectatic lung tissue showed small cystic spaces filled with gelatinous material.

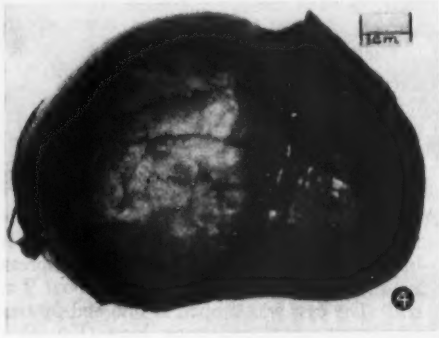
Histological examination of sections taken from the specimen at the junction of the cyst wall and lung tissue cap showed that the lung tissue overlying the large cyst was continuous with the cyst wall (Fig. 5). This cyst was lined by a layer of epithelial cells which in parts had the features of a flattened, pseudo-stratified, ciliated, respiratory epithelium (Fig. 6). The epithelium rested on vascular fibrous tissue in which a few muscle fibres were present.

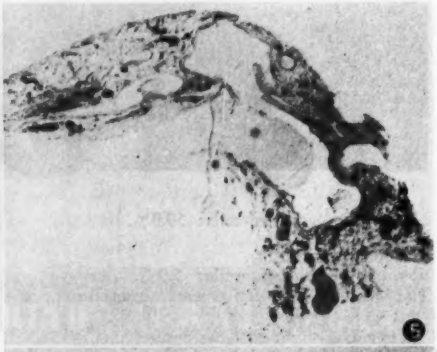
The lung tissue itself was vascular and congested and showed the presence of many cysts which varied greatly in size. Most of these cysts were lined by a typical ciliated pseudo-stratified columnar epithelium. In some of the cysts the epithelium was considerably flattened. Underlying the epithelium was a thin layer of fibrous tissue in which a few smooth muscle fibres were seen, but there was no evidence of a distinct submucosa, nor was any cartilage present. Some of the cysts contained an amorphous material in which a few desquamated cells were present. Many blood vessels were present in the tissue between the cystic spaces and there was also an aggregation of lymphoid tissue in relation to some of the smaller spaces. There was no evidence of dust deposition.

DISCUSSION

Lambert[1] has classified cysts in the thoracic cavity into the following groups:

1. Of parasitic origin, such as hydatid.
2. Dermoid cysts.
3. Teratomata.
4. Arising from embryonal rests of the primitive respiratory system.





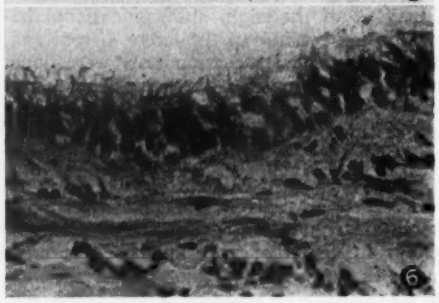


Fig. 4. Photograph of the specimen showing the cyst capped by non-aerated lung tissue.

Fig. 5. Low-power photomicrograph of a section showing the non-aerated lung tissue and the cystic spaces (H & E x 6).

Fig. 6. High-power photomicrograph showing the pseudo-stratified columnar ciliated epithelium lining the cyst (H & E x 70).

5. Arising from the primitive alimentary tract.
6. Arising from the primitive pericardium.
7. Lymphangiomata.

Many synonyms have been applied to congenital cystic conditions of the lungs: the commoner terms such as accessory lung, supernumerary lung, intra- and extra-lobar sequestration, paratracheal cysts and bronchogenic cysts require clarification.

ACCESSORY LUNG

From a review of the literature the conclusion is reached that this term includes many congenital abnormalities of the lung, thus:

(*a*) *With a Bronchial Attachment.*

1. *Supernumerary Lungs.*

(*a*) The tracheal lobe(s) here arise from supernumerary bronchi. These bronchi take origin from the trachea or main-stem bronchus, in addition to those normally present, and are distinct from displaced bronchi, which are displacements of the bronchi normally present.[2, 3] It is this anomaly which is referred to by Henninger and Choy[4] as the 'true accessory lung.'

(*b*) *The Azygos Lobe.* This is an apparently separate portion of the right upper lobe, which is not due to faulty bronchial development but to an abnormal course of the azygos vein. When this vessel curves in a too lateral direction from the prevertebral position to the anterior mediastinum to join the superior vena cava, it splits off the medial portion of the right upper lobe and carries into the fissure so formed a fold of pleura from the posterolateral thoracic wall.

2. *Supernumerary Fissure.* This is an accentuation of the normal bronchopulmonary segments. The commonest is the lingular segment of the left upper lobe becoming separated from the rest of the upper lobe by a fissure.

(*b*) *Without a Bronchial Attachment.*

1. *Intralobar Sequestration.* This is a portion of lung, usually situated in the substance of the left lower lobe, which has no direct communication with the normal bronchial tree. It has an independent arterial blood supply which is usually derived from the lower thoracic or upper abdominal aorta. The venous drainage, however, is to the normal pulmonary system. The sequestrum may consist of one large cyst, but it is frequently multilocular, or it may be composed of a system of branching bronchi which are markedly dilated and filled with mucus.[5]

2. *Extra-Lobar Sequestration.* This is a piece of tissue formed from anomalous, non-functioning pulmonary tissue consisting of cystic bronchi and attenuated air-spaces.[6] It lies outside the normal lung and derives its arterial blood supply from the nearest available source and has its venous drainage into the nearest systemic venous system. It is usually situated above, within or below the diaphragm. This abnormality is often referred to as the lower accessory lobe.[7]

The theory of origin of these sequestrations has recently been reviewed by Boyden.[8]

(*c*) *With a Bronchus Communicating with the Oesophagus.* A few cases have been reported where the accessory pulmonary tissue communicates with the oesophagus instead of the normal bronchial system.[9-11]

PARATRACHEAL CYSTS

These cysts may be of bronchogenic or enterogenous origin, and hence the term does not signify any specific histological type. Paratracheal cysts are cysts of developmental origin which lie in relation to the trachea, and may even be situated in the wall of the oesophagus.[12]

BRONCHOGENIC CYSTS

This term has been widely applied to various cystic masses found in the chest. Some authors term all cysts lined by bronchial epithelium as bronchogenic cysts. Conditions such as intralobar sequestration, extra-lobar sequestration, congenital cystic disease and cystic bronchiectasis have all been included under the term 'bronchogenic cyst.' It should, however, be restricted to developmental cysts of the lung which are lined by bronchial epithelium, but which do not contain alveolar tissue as part of the cyst. These cysts differ from extra-lobar sequestration in this respect, and De Bakey *et al.*[13] consider the presence of normal lung parenchyma about the periphery sufficient reason to call such cysts accessory lobes.

Bronchogenic cysts are usually single; they may be intra- or extra-pulmonary and may or may not have a bronchial or tracheal communication. The blood supply depends upon the situation of the cyst and is usually derived from the nearest available source.

CONCLUSIONS

The case reported is considered to be an extralobar sequestration of the lung for the following reasons:

(*a*) The tissue was situated outside the normal lung tissue.

(*b*) It consisted of non-functioning pulmonary tissue and cysts.

(*c*) The cysts were lined by bronchial epithelium.

(*d*) The venous drainage was into the systemic venous system, viz. the superior intercostal vein.

This case is exceptional, however, because in most of the reported cases of extra-lobar sequestration, the abnormality was found to be in relation to the diaphragm, being either above, within or below it. Here it was related to the left upper lobe. There were no other associated congenital abnormalities which, according to the literature, frequently accompany extra-lobar sequestration.[7]

SUMMARY

A case of extra-pulmonary sequestration of the lung is reported, and some of the relevant congenital abnormalities of the lungs are discussed.

OPSOMMING

Verslag word gedoen oor 'n geval wat om die volgende redes as 'n extralobêre-sekwestrasie beskou word:

(*a*) Die massa was geleë buite die normale longweefsel.

(*b*) Dit het bestaan uit nie-funksionerende longweefsel en kieste.

(*c*) Die kieste was met brongiale epiteel uitgevoer.

(*d*) Die veneuse dreinering was na die sistemiese veneuse-stelsel.

Die meeste gevalle van extralobêre-sekwestrasie wat in die literatuur verskyn het, het 'n noue verband met die diaphragma gehad. Hierdie geval is buitengewoon daar dit in verhouding met die boonste lob van die linker long was. Die ander verwante aangebore abnormaliteite wat, volgens die beskikbare leesstof, extralobêre-sekwestrasie so dikwels vergesel, was nie teenwordig nie.

We wish to thank Dr. A. J. Orenstein, Director of the Pneumoconiosis Research Unit, and Dr. E. H. Cluver, Director of the South African Institute for Medical Research, for facilities to carry out the pathological examination.

Thanks are also due to Dr. I. Webster, Mr. D. I. Adler, and Mr. D. N. Fuller for their advice and interest, Dr. Rautenbach who assisted at the operation and Miss C. E. Campbell for the photographs.

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TRANSIENT PARALYSIS OF THE RECURRENT LARYNGEAL NERVE

DURING BRACHIAL BLOCK

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Brachial block is a valuable method of regional analgesia for operations on the upper limb. The technique has been progressively simplified and to-day the efficacy of the block is assured in almost 100% of cases. The addition of hyalase to the anaesthetic solution has contributed largely to the success of even the occasional local anaesthetist. However, hyalase is so potent in its spreading action that it is advisable to use it sparingly lest the field of analgesia be extended to produce undesirable results.

CASE REPORT

J. E., a European male aged 38 years, was admitted to a hospital on 23 April 1958, having crushed the middle finger of his right hand. He looked older than his age, thin, and unfit, and local analgesia was therefore selected as the anaesthetic method of choice for amputation.

Technique of Brachial Block. At 4.45 p.m. 40 c.c. of 2% Novocaine (to which had been added 1 ampoule of hyalase and 3 minims of

adrenaline 1:1000) were infiltrated in the region of the first rib, just lateral to the sternocleidomastoid muscle. The rib was identified by palpation and the needle introduced directly posteriorly until bony contact was made. It was withdrawn slightly and the solution was injected at this site and a little more laterally. The usual precautions against intravascular injection were taken. After 2 minutes, analgesia was not satisfactory and, to save delay, the tissues around the neck of the metacarpal were infiltrated with Novocaine.

After formal disarticulation of the finger, it was found that the patient had lost his voice. He did not appear hysterical. A few moments' reflection suggested the probable cause, and he was assured that he would regain his voice in an hour or two. In fact, he recovered his voice at about 7 p.m.

Discussion. The right recurrent laryngeal nerve recurs around the first part of the right subclavian artery, medial to scalenus anterior. The muscle is not more than a thumb's-breadth wide, and its lateral edge coincides below with that of the sternocleidomastoid, where the injection was made. The fluid must have spread medially in the plane of the artery, which emerges with the trunks of the brachial plexus from under cover of the lateral edge of scalenus anterior, to reach the nerve. It seems possible that the wide diffusion of the fluid may have been responsible for the unusual failure of the block to be rapidly effective.

CONCLUSION

When performing a brachial block, it appears advisable to use hyalase sparingly (say, 2 or 3 minims of the solution as made in the ampoule) in order to minimize the possibility of recurrent laryngeal nerve palsy. The possibility is probably more remote on the left side, where the nerve recurs around the aortic arch in the thorax and then ascends in the tracheo-oesophageal groove close to the midline.

Recurrent nerve palsy as a complication of brachial block does not appear to have been previously reported.

OPSOMMING

By die uitvoering van 'n bo-arm-blokkade skyn dit raadsaam te wees om hialase spaarsaam te gebruik om die moontlikheid van wederkerende larinkssenuweeverlamming so klein as moontlik te maak.

Die moontlikheid is waarskynlik kleiner aan die linkerkant, waar die senuwee weer voorkom rondom die hoofslagaarboog in die borskas, en dan digby die middellyn in die lugpyp-slukderm-groef opstyg.

Wederkerende senuweeverlamming as 'n verwikkeling van bo-arm-blokkade is skynbaar nie tevore gerapporteer nie.

CYANOSIS IN INFANCY

AN OUTBREAK OF METHAEMOGLOBINAEMIA IN A NURSERY

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It is important to outline briefly the processes of respiration so that the mechanisms involved in the production of cyanosis are more readily understood.

Pulmonary ventilation involves the uptake of oxygen and the elimination of carbon dioxide. Then the oyxgenated blood is transported to the tissues, where utilization of oxygen and the discharge of carbon dioxide occur. For the present discussion the uptake of oxygen and the discharge of carbon dioxide may be considered together.

The oxygen uptake is first under the control of the respiratory centre which may be affected by numerous intracranial lesions. It may also be impaired by obstructive influences along the whole respiratory tract; in addition, the lung parenchyma may be diminished by external influences pressing from without.

If there are no disease processes in the lungs the oxygen uptake may be diminished by poor cardiac function, as in congestive cardiac failure causing stasis in the lungs or in certain right-to-left shunts where the unoxygenated blood is mixed with the oxygenated blood, with resulting cyanosis.

The next phase is the incomplete utilization of oxygen by the tissues or the stagnation of the blood in the periphery with accumulation of carbon dioxide, the production of cyanosis being due to poor capillary tone or venous stasis.

In addition, the oxygen exchange may be affected by changes in the carrying capacity of blood, as exemplified by a change of haemoglobin to methaemoglobin or sulphaemoglobin.

If the cyanosis is pulmonary in origin it will be improved by oxygen inhalation; in the

cardiac case, however, oxygen will not help; nor will the colour due to methamoglobin be improved. Another point is that pulmonary cyanosis is alleviated by crying, whereas it is aggravated when the cause is cardiac.

The various factors are tabulated in Table 1.

TABLE 1: CLASSIFICATION OF CYANOSIS

A. *Peripheral Causes.*
B. *Central Causes.*
1. Intracranial.
2. Lung.
3. Heart.
C. *Pigmentary Causes.*
Methaemoglobin.

PERIPHERAL CYANOSIS

Peripheral cyanosis is distinguished by the fact that arterial oxygenation is above 91%. It is usually manifest in hands and feet and not in the mucous membranes. This is a very frequent occurrence in the new-born and is a completely benign finding which usually disappears within 24–48 hours. Peripheral cyanosis may also be a finding in severe toxic states where the toxicity results in hypotension and peripheral stasis. Severe inflammatory lesions, in the brain and lungs, are 2 good examples. The cyanosis is not only central but also peripheral. A good example showing the overlap is a recent case of a 12-day infant seen by the author, who suddenly developed pneumonic signs. In a few hours the anterior fontanelle was bulging but there was no neck rigidity. The infant was toxic and cyanosed. A lumbar puncture showed cerebrospinal fluid loaded with pus cells and a culture yielded pneumococci. Cyanosis in this case could be attributed to peripheral, lung and intracranial causes.

INTRACRANIAL LESIONS

Cyanotic attacks due to disturbances of the central nervous system are usually characterized by a delayed onset of breathing at birth; periodic bouts of apnoea, cyanotic spells, hypotonicity or hypertonicity. At times there is fullness of the anterior fontanelle, neck retraction and somnolence. The sucking reflex is poor and the Moro reflex may be absent. Twitchings or actual convulsions may be a feature.

Intraventricular haemorrhage is common in the premature. The onset of cyanosis may be sudden, usually with a fatal termination. In full-term infants the bleeding is usually subdural. A history of trauma or difficult delivery is often not elicited.

Hypoxia may be produced by premature separation of the placenta, compression of the umbilical cord, prolonged labour or hypotension in the mother. If the hypoxia is severe the result may be a still-born infant. If less severe, the signs of intracranial haemorrhage may be manifest. The pathology consists of minute haemorrhages in the brain and varying evidence of aspiration of amniotic fluid into the lungs.

Congenital anomalies of the brain may cause similar findings. Besides obvious malformations such as hydrocephalus and anencephaly, gross anomalies may only be detected *post mortem.*

Inflammatory lesions will usually be evident a few days after birth. Encephalitis, often due to the Coxsackie virus, may occur and sometimes proves fatal. In the cases of meningitis that occur in the new-born period the organism is either *B. coli,* staphylococcus, streptococcus, or pneumococcus. Vigorous and early treatment is essential to save life and prevent irreparable damage, or subdural haemorrhage or effusions. The chief intracranial lesions are tabulated in Table 2.

PULMONARY CAUSES

Pneumonia may occur as a complication of aspiration, post-maturity or hyaline membrane disease. In these cases it can only be diagnosed *post mortem.*

In a small number of cases pneumonia may be a primary disease acquired *in utero,* or in early post-natal life. The initial symptoms may be listlessness, refusal to feed and loss of weight. Cyanosis may be marked, although fever and cough may be slight.

TABLE 2: INTRACRANIAL LESIONS

1. *Haemorrhage.*
(a) Intraventricular.
(b) Subdural.
2. *Hypoxia.*
3. *Congenital Abnormalities.*
4. *Inflammatory Lesions.*
(a) Encephalitis.
(b) Meningitis.

The physical findings in the lung may be slight although the respiratory rate is very rapid and rib retraction may be present. The organisms are usually pneumococci, but Friedlander's bacillus and staphylococci may be responsible. In the latter 2 instances the pneumonia is often complicated by lung abscesses and pyopneumothorax. These complications are only proved on X-ray examination.

The distinguishing features from giant cell pneumonia, virus pneumonia, cytomegalic inclusion disease, fungus disease and haemorrhagic lung disease are only differentiated histologically.

PULMONARY HYALINE MEMBRANE

This condition now appears to be generally accepted as a definite entity. The literature was recently reviewed by Tran-Dink-De and Anderson.[1] It is due to a layer of mucoprotein covering the alveolar lining and resulting in marked respiratory distress. The origin of the mucoprotein is controversial. Suggested theories are aspiration of amniotic fluid, transudation of protein from pulmonary capillaries, cardiac failure or oxygen intoxication.

It occurs mainly in premature infants, infants from diabetic mothers and babies delivered by caesarean section. The infants are usually normal at birth but within 1–3 hours there is marked respiratory distress, increasing cyanosis, laboured respiratory effort with marked intercostal retraction but poor air entry. On X-ray there is a fine granular reticular pattern. Death usually occurs within 24–48 hours. At necropsy the lungs are red and non-crepitant, and the histological sections show diffuse hyaline membrane covering all the alveoli. If death is delayed, additional findings may be emphysema and infection.

PNEUMOTHORAX AND OBSTRUCTIVE EMPHYSEMA

These 2 conditions are considered together because they have one thing in common, i.e. they both show sudden onset of cyanosis and also because active surgical intervention may be life-saving. Pneumothorax usually follows a mediastinal emphysema which may occur spontaneously or be due to active artificial respiration. If the pneumothorax is large then tension can occur and there is marked distress which can be relieved by decompression. In the case of obstructive emphysema one lobe is involved and the cyst may become very large. It causes a shift of the mediastinum and often on X-ray it is found to extend to the opposite side. An X-ray will easily differentiate these two conditions.

EXTRA-PULMONARY CAUSES

Here may be included a number of conditions which may produce cyanosis. Although there is no actual pulmonary disease, the lungs are involved indirectly.

Diaphragmatic hernia is an example. It may be congenital. Cyanosis is a prominent feature, with evidence of dullness and abnormal signs in the chest associated with a scaphoid abdomen. Surgery must be instituted before the abdomen becomes distended with air, otherwise it becomes difficult technically.

Tracheo-oesophageal fistulae, another example cause mucus in the mouth, and periodic bouts of cyanosis associated with feeding. It can be confirmed by a Lipiodol swallow. Operation is life-saving and often successful. Another rare condition which may show periodic bouts of cyanosis or swallowing difficulty is a vascular anomaly of the great vessels. The two most frequent are double aortic arch and an aberrant right subclavian artery. In these cases operation can usually be deferred until the infant is much older.

The various lung conditions are classified in Table 3.

TABLE 3: LUNG CAUSES

1. *Pneumonia.*
2. *Hyaline membrane.*
3. *Pneumothorax and Obstructive Ephysema.*
4. *Extra-Pulmonary Causes.*
   (a) Diaphragmatic hernia.
   (b) Tracheo-oesophageal fistula.
   (c) Vascular anomalies.

HEART DISEASE

Heart pathology as a cause of cyanosis is of ever-increasing importance because of the greater skills which the cardiac surgeon has evolved in tackling very complicated cases of congenital heart disease.

Medical treatment is also most effective in many cases of congestive cardiac failure, which must be relieved so that the infant can be rendered fit to undergo cardiac surgery, if indicated.

In the next few years, when the heart-lung machine has been perfected, many cases of congenital heart disease will be repaired effectively so that an accurate diagnosis of heart disease is imperative.

In infancy, cyanosis may be considered in 2 broad groups. In the first group cyanosis is but a symptom of congestive heart failure. The underlying pathology may be infective, metabolic or congenital. Symptoms may be absent until cardiac failure sets in. In the second group cyanosis is the predominant feature. The cyanosis in all cases may be due to inadequate oxygenation, peripheral stasis or a severe shunt.

In all cases a complete history and a physical examination are essential and may be diagnostic. Routine aids are an electrocardiographic tracing which includes the standard leads and also unipolar limb and chest leads. The electrocardiogram may be pathognomonic, but in the majority of instances will indicate either left or right heart strain. The second essential investigation is a chest X-ray, which will indicate cardiac enlargement or specific chamber enlargement. It also shows the vascular state of the lung fields. Cardiac catheterization and angiocardiography is of limited value in the newborn period.

Electrocardiography. The infant in the newborn period shows right ventricular prominence, which gradually changes over to the adult pattern. In most cases it is complete at 9 months.

Left ventricular hypertrophy may be apparent in the standard leads as shown by a tall R in lead I and a deep S in lead III. Another finding may be a tall R in leads II and III. In the unipolar chest leads the findings are a deep S in V1 and V2 and R high in V5 and V6. A depressed ST or inverted T in V5 and V6 is conclusive evidence of left ventricular hypertrophy.

Right ventricular hypertrophy in standard leads may show a deep S in lead I and a tall R in lead III. Also a deep S in leads I, II and III is suggestive. In the unipolar leads there may be a tall R in V1 and V2 as well as an inverted T in V1 and V2. Another helpful point is an upright T in V1.

TABLE 4: CLASSIFICATION OF HEART DISEASE

Group A

Presenting Feature: Congestive Cardiac Failure

1. Acute aseptic myocarditis.
2. Paroxysmal tachycardia.
3. Coarctation of the aorta.
4. Patent ductus arteriosus.
5. Congenital mitral stenosis.
6. Erythroblastosis.

Group B

Presenting Feature: Cyanosis

1. *Left Ventricular Dominance.*
   (*a*) Tricuspid atresia.
2. *Right Ventricular Dominance.*
   (*a*) Complete transposition of the great vessels.
   (*b*) Incomplete transposition of the pulmonary artery with a completely transposed aorta (Taussig-Bing).
   (*c*) Completely transposed pulmonary veins.
   (*d*) Incomplete transposition of the aorta with the pulmonary artery from the right ventricle (Fallot).
3. *Combined Heart Strain.*
   (*a*) Truncus arteriosus.
   (*b*) Single ventricle.

Acute Aseptic Myocarditis, a terms coined by Blattner,[2] resembles the original Fiedler's myocarditis.[3] The condition may occur in epidemics and in a number of instances the Coxsackie virus[4] has been implicated. There is an acute onset of cardiac failure, pyrexia, cyanosis, cardiomegaly and electrocardiographic changes. This condition responds well to digitalis. Digitoxin is a stable preparation and can be given in doses of 0.06 mg. per kilo per 24 hours.

This disease must be differentiated[5] from (*a*) endocardial fibro-elastosis; (*b*) medial necrosis of the coronary arteries; (*c*) aberrant left coronary; (*d*) glycogen storage disease. All the above conditions have cardiomegaly, electrocardiographic changes and absence of murmurs, but each has specific changes: all show symptoms a little later in life. A benign type of pericarditis[6] due to the Coxsackie virus must also be considered.

Paroxysmal Tachycardia is a rare condition which occurs most frequently in infancy, characterized by a sudden onset. If the symptom persists, congestive cardiac failure ensues. Nearly all cases are of the supraventricular type and respond dramatically to digitalis. In the ventricular type quinidine or procaine may be beneficial.

Coarctation of the Aorta.[7] In most cases this entity remains asymptomatic and is successfully repaired surgically in childhood. In a few instances it may start in infancy with congestive cardiac failure and cardiomegaly. Diminished femoral pulses are diagnostic. If a patent ductus arteriosus is present distal to the coarctation, the femoral pulses may be normal, but the feet are more cyanosed than the hands. Cardiac failure can be reversed with digitalis. Surgical intervention should be postponed as long as possible.

Patent Ductus Arteriosus may present in infancy with cardiac failure. This is either due to a large defect or in some instances there may be a primary pulmonary hypertension with a reversed blood flow. The murmur is atypical at this age and the electrocardiogram may show right ventricular strain.

Congenital Mitral Stenosis may show evidence of cardiac failure with a typical murmur. Early mitral valvotomy may prolong life.

Erythroblastosis.[8] In severe cases of erythroblastosis due to Rh incompatibility the infant may at first be cyanosed with evidence of failure, marked oedema and ecchymoses, the intravenous pressure being raised above 10 cm. of water. Within a few hours jaundice will be marked and superadded. Early replacement of blood is life-saving.

Tricuspid Atresia[9] presents with marked cyanosis at birth. Cardiac failure may be present. The electrocardiogram shows marked left ventricular strain and right auricular dilatation (P. pulmonale). On X-ray the heart is usually enlarged with a straight right border because of the absent right ventricle. Survival is only possible if associated shunt defects are present. A palliative operation may be either a Pott's or a Blalock procedure.

Complete Transposition of the Great Vessels is incompatible with life unless an associated shunt is present. Cyanosis is marked from birth. Cardiomegaly occurs within a few weeks. Right ventricular strain is always present. Murmurs are not of any signficance. Veins on the scalp may be prominent. With the new heart-lung machine surgical repair will be possible.

Incomplete Transposition of the Great Vessels.[10] (Taussig-Bing Anomaly). There is a transposed aorta and an over-riding pulmonary artery, with an associated septal defect. Cyanosis is marked from birth. Survival is a little longer than in the fully transposed type.

Completely Transposed Pulmonary Veins show marked cyanosis at birth. The X-ray shows a characteristic supracardiac shadow.

Fallot's Tetralogy is characterized by pulmonary stenosis, an over-riding aorta, ventricular septal defect and right ventricular hypertrophy. This is the commonest cause of cyanosis in children but only a third of the cases have cyanosis in infancy. The pulmonary artery may be absent on X-ray and the pulmonary vasculature is diminished. The heart is not enlarged. The 3 operations which can be performed are Blalock, Pott's and Brock procedures.

Persistent Truncus Arteriosus. The degree of cyanosis depends upon the size of the pulmonary vessels. A machinery murmur is common, often down the left sternal border. X-rays show combined left and right ventricular enlargement with an absent pulmonary artery. If cyanosis is marked a shunt procedure may be palliative.

Single Ventricle. If pulmonary artery stenosis is associated with a single ventricle, the cyanosis will be marked; if the pulmonary vessel is patent, cyanosis may be strangely absent. The unipolar chest leads often have a bizarre appearance in that they all resemble one another. The pulmonary vasculature will be diminished.

The cases which have been stressed are those in which cyanosis is a feature in early infancy. Cases which may develop cyanosis later have not been discussed although this may occur in some of the instances quoted.

BLOOD PIGMENT: METHAEMOGLOBIN

A CASE REPORT

In June 1957, 4 infants in a maternity block of a country hospital were all acutely ill. The respirations and heart rates were rapid but the temperature was normal.

A likely diagnosis in the circumstances was methaemoglobinaemia. Therefore a search was made for recently marked linen. Napkins freshly marked in large print were found on all 4 infants. There was also a strong aromatic odour in the room aggravated by the heating system.

The first infant, although in an oxygen tent, still had a grey colour. His respiratory rate was 70 and the pulse rate 140 per minute. His breathing was deep and regular. The breath sounds were clear. There was no cardiac enlargement. No murmurs were heard. The temperature was 98° F. Except for the generalized cyanosis over the whole body, the remainder of the examination was essentially within normal limits. The infant appeared to be critically ill.

Three other infants seen at the same time were cyanosed to a lesser degree. The cyanosis had been present for 15 hours. In one case the cyanosis had greatly lessened. Two further cases developed 24 hours later. Further details of the infants can be seen from Table 5.

TABLE 5

Baby	*Age in Days*	*Birth Weight*		*Present Weight*	
		Lb.	*Oz.*	*Lb.*	*Oz.*
G.	2	8	1	8	—
W.	4	7	3	6	13
N.	2	7	12	7	12
B.	8	5	5	5	2
V.	1	8	7	8	7
P.	20	3	4	3	2

None of the babies had received any medicine nor had the nursery been recently fumigated. The blood from Baby G (who was the most cyanosed) was collected for testing before treatment was administered.

Findings:		
	Haemoglobin	16·9 %
	Oxyhaemoglobin	14·9 %
	Methaemoglobin	1·9 %
	Sulphaemoglobin	0·067%

Powdered methylene blue was obtained from an emergency dispensary and 60 mg. were weighed out. The powder was autoclaved and then dissolved in 20 c.c. triple distilled water. The first 4 infants were given 2 c.c. (6 mg.) of the mixture intravenously. The next day the fifth was given 2 c.c. and the 6th, a premature infant, only received 0.5 c.c.

In the first 4 cases the cyanosis completely disappeared in about 45 minutes, although the respiratory rate continued to be rapid the results were equally dramatic.

The diagnosis of methaemoglobinaemia was confirmed by the blood examination and the dramatic response to methylene blue.

HISTORY OF METHAEMOGLOBINAEMIA

In 1886 Rayner[11] first described 17 cases of methaemoglobinaemia due to marking ink on diapers. In 1920 Ewer[12] reported two cases due to nitrobenzine inhalation in the German literature. In 1928 Stevens[13] reported a further case due to nitrobenzine poisoning. This substance was apparently used to fumigate the cot. In 1947 Wallace[14] reported a case due to the ingestion of bismuth subnitrate. The subnitrate apparently was changed in the bowel to nitrite. In 1949 Kagan *et al.*[15] reviewed the literature and found 5 deaths in 63 cases. They added 9 cases of their own.

In 1949 Donahoe[16] in the United States reported 5 cases due to the drinking of water from a well which contained a large amount of nitrates. In 1955 Goluboff and MacFadyen[17] reported a case in a 3-month-old infant suffering from a severe attack of eczema and who was treated with a coal-tar preparation over a large area of the skin.

In 1956 Dine[18] reported the 2 youngest cases of congenital haemoglobinaemia, aged 4 days and 18 days, respectively. Both improved on intravenous methylene blue and ascorbic acid.

The various factors which may cause methaemoglobinaemia are tabulated in Table 6.

TABLE 6: CAUSES OF METHAEMOGLOBINAEMIA

Familial.
Aniline Dyes
(a) Contact;
(b) Inhalation.
Nitrobenzine.
Bismuth Subnitrate.
Nitrates in Drinking Water.
Tar Ointment Application.
Potassium Chlorate.
Acetylsalicylic Acid in Large Doses.

MECHANISM

Aniline ($C_6H_5NH_2$) is a colourless oily liquid. It has an aromatic odour and a burning taste. It is derived from the reduction of nitrobenzine. It is slightly soluble in water, but very soluble in alcohol, ether, benzine or chloroform. Intoxication may occur either from cutaneous absorption or inhalation. In the present cases inhalation may have been a factor, as the nursery was very warm and humid.

If aniline is mixed with blood the change to methamoglobin is very slow. It is therefore thought possible that the aniline dye is changed to a more active oxidizing agent such as paraminophenol or hydroxyphenyl-hydroxylamine. The haemoglobin is apparently oxidized to methaemoglobin, the ferrous iron being changed to ferric iron. The oxygen capacity of the blood to give up oxygen to the tissues is reduced. Methylene blue in large doses also converts ferrous iron to ferric iron but in small doses it reverses the process. Ascorbic acid has a similar action but acts much more slowly.

Finch[19] states that a level of 1.5 g. of methaemoglobin can produce visible cyanosis. Barcroft *et al.*[20] found that in congenital methaemoglobinaemia there was a defect of co-enzyme factor in the erythrocyte. This enzyme, also known as diaphorase 1, is an essential link in the chain of oxidation enzymes which maintain the haemoglobin in a functional state. If deficient, the haemoglobin is changed to methaemoglobin. This change is rapidly reversed with methylene blue. It is possible that the aniline dye actually diminishes the diaphorase 1.

Betke[21] found that foetal haemoglobin is more easily converted to methaemoglobin than is adult haemoglobin. This may be the reason that newborn infants are so susceptible to this condition.

Methaemoglobin must be differentiated from sulphaemoglobin (which may occur with certain drugs, but is not a real problem in infancy). This can be done by comparing the bands on the spectroscope. Methaemoglobin disappears from the blood on the addition of sodium cyanide or sodium hydrosulphite but the sulphaemoglobin is not affected by these reagents.

SYMPTOMS OF METHAEMOGLOBINAEMIA

The onset is usually acute with the appearance of a grey colour which does not change when the infant is placed in an atmosphere of oxygen. The respirations are rapid but regular. There is a usually marked loss of appetite but in these cases the nursing staff notice that the infants are well. More severe symptoms which may result are tremor, convulsions, tinnitus and paralysis. In large doses there may be heart block or other cardiac arrhythmias. Rare findings are haematuria, abdominal distension and jaundice. Death may follow in untreated cases.

TREATMENT

The best treatment is 1% methylene blue, given intravenously in a dose of 1–2 mg. per kilo body weight. The result is most dramatic. If a vein cannot be found, the methylene blue

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can be given orally in double the dose. In this case the change in colour is much slower. Ascorbic acid has also been given with results comparable to those of oral methylene blue.

SUMMARY

An outbreak of cyanosis due to methaemoglobinaemia stimulated the author to discuss the various causes of cyanosis which may occur in early infancy.

The mechanism of cyanosis is briefly outlined and a broad classification set out. This shows that cyanosis can be grouped into 3 categories: peripheral, central and pigmentation of blood.

The *peripheral* causes are usually benign and are briefly mentioned. The *central* causes are intracranial, pulmonary and cardiac. The various conditions which fall into these 3 categories are discussed in detail and tabulated.

The report of 6 cases of methaemoglobinaemia in a nursery are discussed. The history, symptoms and treatment of methaemoglobinaemia are reviewed.

In the outbreak mentioned the cause was traced to the aniline dye in recently marked napkins.

OPSOMMING

'n Uitbreking van sianose in 'n kinderafdeling wat aan methemoglobinemie te wyte was, het die skrywer aangespoor om die verskillende oorsake van die sianose wat tydens die suigelingsjare voorkom, te bespreek.

Die meganisme van sianose word kortliks geskets, en 'n breë klassifikasie word verstrek. Dit toon aan dat sianose in drie groepe saamgevat kan word—randstandig, sentraal, en pigmentasie van die bloed.

Die randstandige oorsake is gewoonlik goedaardig, en word net in die verbygaan genoem. Die sentrale oorsake kan weer in intrakraniale, pulmonale en hart-afdelings ingedeel word, en die verskillende toestande wat onder hierdie afdelings ressorteer, word breedvoerig bespreek en getabelleer.

Die verslag oor 6 gevalle van methemoglobinemie in 'n kinderafdeling word bespreek. Die geskiedenis, simptome en behandeling van die methemoglobinemie word in oënskou geneem.

Daar is vasgestel dat bogenoemde uitbreking veroorsaak is deur die anilienverfstof waarmee luiers kort tevore gemerk is.

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YELLOW FEVER AND THE RECEPTIVE AREA OF THE WITWATERSRAND

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The epidemiology of yellow fever in Africa is far from clear. Many factors still elude investigators. A passage from de Meillon's report[1] *The Threat to Ngamiland of the Introduction of Some Tropical Diseases* can be quoted to illustrate this:

'The epidemiology of yellow fever in Southern Africa is puzzling and has not been worked out. The facts at the moment are:

1. Up to as much as 10% of the indigenous population show immunity to the disease;

2. Potential invertebrate vectors are known to occur;

3. No reservoir hosts have so far been found.

Two explanations of the present position suggest themselves:

1. That the disease is mildly endemic, that reservoir hosts are absent and that epidemics do not occur because of the long break in the transmission season, it being known that the usual vectors are absent for several months during the year.

2. That active virus is being introduced at intervals by itinerant labour from the north; that this has not resulted in widespread infection because its introduction is erractic and rare and because of the nature of the virus, transmissible for only short periods, and that so far it has not spread to any reservoir hosts, e.g. monkeys.

At the moment sufficient data have not been collected to decide between (1) and (2), but such evidence as exists points strongly to (2). If this is so, then the possibility of circumstances arising which will favour an epidemic and the establishment of the virus in reservoir hosts is not remote. It has already been pointed out that 'conditions prevailing in Ngamiland and Barotseland have much in common with those described in the Anglo-Egyptian Sudan shortly before the outbreak of the 1940 epidemic.'[2]

In any case it would appear that the Bantu shows a relatively milder reaction to the disease than do Europeans and others. The spread of the virus southwards to the Union would depend on either a slow penetration into new receptive areas or on the rapid transport of infected mosquito vectors or infected human beings. In the first case one could expect a gradual extension of the areas southwards from which human immunes could be recovered, while in the second case explosive but isolated epidemics at the terminals of travel routes in receptive areas could be expected.

However, apart from the constant discovery of human immunes recovered from the silent yellow fever areas in routine mouse protection tests, and apart from a few explosive outbreaks, such as the epidemics which took place in the Nuba mountains (1940)[3] and in other areas in northern Central Africa where natural immunes were already proved to exist in the years preceding the epidemics, no evidence of the virus moving into the Union has yet come to hand. This movement, or lack of movement, of virus is very puzzling. Yet there is a constant threat to all towns and cities on the Southern Subcontinent where the vectors of the disease are known to exist, as immunes are discovered in new work being done further south.

In one area in Kenya where extensive research has already proceeded on some aspects of the problem, it is felt that the infection is of long standing and not merely of recent introduction associated with modern travel facilities.[4] But it may also be pointed out that infection is retarded there in reaching the human population by the common occurrence of artificial immunity given to about 7% of the population with the inoculation of 17D yellow fever vaccine. Again, apart from the broader aspects of the failure of yellow fever virus to establish itself in the East, there is the specific case of travel contact between the East Coast of Africa and the ports of Asia. The contact is maintained by Arab dhows, which have plied between Kenyan and Asian ports for years. The classical vector of yellow fever—*Aedes aegypti*—has been shown to breed on board the dhows, but never have any Asian outbreaks of yellow fever occurred.

Work done in Southern Africa has shown human immunes (presumably not artificially induced) as far south as the Zambesi, far away from the known endemic areas and in the absence of the usual environment of tropical forests containing monkey populations. It is not known how these immunes have come about.[5]

The precautions laid down in the International Sanitary Regulations are observed in regard to all passengers and air transport plying between endemic yellow fever areas and the sanitary airports of the Union,[7] yet thousands of illegal immigrants cross the borders of the Union annually, many coming from the classified yellow fever areas. In spite of this 2-way pedestrian traffic through endemic areas and receptive areas, no case has been reported to date within the boundaries of the Union, and all monkeys examined have proved negative. It appears that a more methodical breakdown of the African yellow fever zone, as defined in international literature, should take place into areas as is done in South America in order to make it easier to gauge the true degree of risk.

Native labour from tropical areas (as distinct from Mozambique) is recruited for industry in the Union from Southern Rhodesia, Northern Rhodesia, Nyasaland, Bechuanaland, Northern South West Africa, Angola and as far north as Tanganyika.[8] All types of transport are used to move this labour force to and from the Union, while considerable numbers of individuals walk hundreds of miles to cross the borders illegally. By these means more than 60,000 labourers move backwards and forwards over the borders of South Africa.

Some of the farthest points north from where itinerant labour is gathered for movement south to the Union are at Grootfontein and Runtu in South West Africa, Siwelewele, Sikango, Kalabo, Nguvu and Chavuma in Northern Rhodesia bordering on Angola, and

Fort Hill and Karonga in Nyasaland. The labour organization of the South African gold mines has established stations and rest camps throughout Barotseland and Nyasaland with permanent depots at Maun, Mohembo and Francistown. Francistown is the gathering point where labour is collected before embarking on the last leg of the journey to Johannesburg, where recruits are allocated to the various gold mines along the Witwatersrand and in the Orange Free State.[8]

The endemic yellow fever area in Africa has been delineated by the Yellow Fever Panel of the World Health Organization in 1949 as recommended by UNRRA in 1944:[9, 10]

'All the territories lying between parallel 15° N and 10° S excluding Tanganyika, with the addition of the territory to the south of Barotseland between 23rd and 25th east meridians of longitude and down to the 21st parallel of south latitude.'

Mahaffy *et al.*[11] have found a percentage of positive reactors in Nyasaland whose inclusion will probably be discussed at the next session of the WHO expert panel.

A comparison of these endemic areas with those from which tropical labour comes, shows on the west that the Native labour sources of South West Africa and Angola (except the extreme north), which are open to the Union, lie outside the general delineation, but that many of the transport routes used from there as well as some of the recruiting areas in Northern Bechuanaland and Southern Barotseland fall into the separate delineation of 23rd-25th meridians east and 21st parallel south latitude. On the east, Nyasaland, which supplies a large proportion of the labour requirements, has still to have its position clarified. Also the most northerly stations in Nyasaland act as collecting points for labour from territories even further north. Accelerated travel by air could result in the introduction of virus into receptive areas if sufficient precautions were not observed. Yellow fever receptive areas are defined[5] as areas in which yellow fever does not exist but where conditions would permit its development if introduced.[12] The conditions which would permit the outbreak of yellow fever in receptive areas are the introduction of the virus in mosquitoes or in Man, the presence of a concentrated non-immune population, and the presence of proven vectors of the virus.

The African vectors of yellow fever with their distribution have recently been listed by de Meillon as follows:[13]

(*a*) *Proven Vectors*:
Aedes (*s*) *aegypti*: Widely distributed except in the Western Cape.
Aedes (*s*) *simpsoni*: Occurs in the eastern subtropical corridor from the Northern Transvaal to East London.

(*b*) *Suspected Vectors*:
Aedes (*s*) *vittatus*: Widespread in northern South West Africa, Transvaal and Natal, Swaziland and Northern Bechuanaland.
Aedes (*s*) *metallicus*: Widespread in the Northern Transvaal and the subtropical belt.
Diceromyia taylori: Found once in Northern Zululand.
Taeniorhynchus africanus: Occurs on the Natal coast, at Onderstepoort and in Northern Bechuanaland.

In addition to these there are 6 other species not mentioned here which have a doubtful significance in that they are merely suspected vectors.

The surveys carried out by the staff of the Plague Research Laboratory of the South African Institute for Medical Research, in the years 1951–1953,[14, 15] on the Witwatersrand and its vicinity, showed the presence of at least 2 proven vectors in that area. *Aedes* (*s*) *aegypti*, the classical vector of yellow fever, was found extending from the Far East Rand to the Far West Rand. *Aedes* (*s*) *metallicus* was only recovered from one place on the East Rand. *Aedes* (*s*) *aegypti* was recovered from gold mining areas at Springs, New Modder and Luipaards Vlei and also from the urban areas of Springs, Benoni, Germiston (airport) and Krugersdorp, as well as from the peri-urban areas north of Johannesburg, Fountains, Irene and at Heidelberg.

The Stegomyia mosquitoes were shown by these surveys to be sparse and to be affected by the severe climatic changes experienced on the Witwatersrand. They appeared to be difficult to find in poor entomological years, such as 1951, but fairly common under good weather conditions such as were experienced in 1952-1953. It seems that they penetrate back into unfavourable areas (from which they die out during adverse conditions) from areas situated along the northern fringe of the Witwatersrand such as Irene and Fountains and other neighbouring peri-urban areas.

A consideration of the hydrographical features of the Witwatersrand shows that most mosquito breeding proceeds in container-like places associated with domestic dwellings.

The mosquito population of this area is not directly proportional to the water surfaces found there. A classification of the significant breeding places found in surveys is given in Table I.

Only about 20% of all breeding took place in collections of water and streams situated away from human habitations, even though these water surfaces are enormous when compared with the more limited water surfaces from which most breeding came, i.e. drains, rain water tanks, fish ponds and surface drains. Together these latter water surfaces produced 80% of all breeding, open drums of water standing in gardens having the most prolific breeding, followed in order of importance by rain water tanks, fish ponds and compound surface drains, while open drums near compounds gave rise to only very little breeding.

Small container-types of breeding places were not found often, except at refuse deposit sites, which are very limited in number and strictly controlled by the Municipal Health Authorities. They do not form any problem, particularly as the intervals between showers of rain are usually long enough to ensure complete evaporation of their water contents.

Plants and trees do not offer many breeding facilities on the Witwatersrand, as the usual large axiled plants and tree holes associated with this type of breeding are not found here.

Most water courses and dams fed by water courses on the southern slopes of the watershed are contaminated by the rainwater runoff from the gold mine sand dumps and slimes dams. The only collections of water not having a trace of contamination are pans which fill up with rainwater and which are not fed by surface streams. Some minor streams originating from sewage purification plants, etc. may not come into contact with mine sand leachings for some distance, but eventually they discharge into other bodies of water bearing away sand. In addition, many millions of gallons of water are pumped up from the underground workings into surface dams, the overflow from these dams flowing into the streams. On the northern slopes of the watershed there are few mine sand dumps and the water is therefore less contaminated with sand leachings. The sand leachings and the underground water combine to lower the pH content of all surface water with which they come in contact. These contaminated water surfaces having a high degree of acidity are extensive on the southern slopes, but support little aquatic life, and the vegetation in contact with these waters is also different from that bordering on uncontaminated water. More vertical growth and grosser plants characterize these places, thick high reeds being common in some areas, while grass with thick upright stems grows sparsely in others.

Table 2 shows the pH values of water contaminated with water pumped from underground workings and with mine dump sand leachings; with little or no contamination and from rainwater tanks in their relation to mosquito breeding.

In the past, in campaigns against yellow fever and in the struggle against its introduction into new areas, anti-mosquito measures have played the major part.[16] Measures against mosquitoes in the Panama Canal zone were largely responsible for the stamping out of the disease there, and in South America control has been largely brought about by the reduction of the *Aedes aegypti* index below 1% in all towns and ports. In 1950 the WHO Expert Committee on yellow fever stressed the desirability of similar eradication programmes being undertaken in Africa.[17]

Should the threat of yellow fever ever become real to the Witwatersrand, control measures could be applied along the classical lines of species sanitation. Many such examples of mosquito vector control programmes have been recently carried out, the classical one being the eradication of *Anopheles gambiae* from Brazil, and more recently the eradication of all mosquitoes from Cyprus. In the problem of mosquito control on the Witwatersrand almost complete eradication of the vector species is feasible and economical because of the favourable geographical and climatological conditions present, and most certainly com-

TABLE 1: MOSQUITO BREEDING FOUND (EXPRESSED AS % OF TOTAL)

A. *Away from Human Habitation*				B. *In Association with Human Habitation*			
Collections of Water				*Town or Mine Dwellings*			*Compounds*
Streams	*Dams or Lakes*	*Pans*	*Vleis*	*Drains*	*Rain Tanks*	*Ponds*	*Drains*
10	5	4	1	29	17	16	18

TABLE 2: MOSQUITO BREEDING FOUND (EXPRESSED AS % OF TOTAL)

pH Value	*Locus*	*Source of Contamination*	*Mosquito Breeding Present*
7·6	Rand Water Board	Tap water	
		A. Water with Mine Sand Leachings	
6·6	Roodepoort C.M.R.	Stream sand distant	−
6·5	Roodepoort Rand Leases Yacht Club	Dam sand distant	−
6·3	Randfontein Delmas Milling Co.	Stream sand distant	−
6·2	Randfontein Estates	Stream sand distant	−
5·8	Johannesburg, Rosherville	Dam sand near	−
5·2	Krugersdorp, Rietvlei	Stream sand near	−
5·0	Boksburg Lake	Lake much sand	−
4·5	Canada Dam	Dam much sand	−
4·4	Germiston Lake	Lake much sand	−
3·9	Brakpan, G.G.M.A.	Stream much sand	−
3·6	Wemmer Pan	Dam much sand	−
3·5	Springs, Klein Blesbok	Spruit much sand	−
3·4	Boksburg, E.R.P.M.	Stream, sand and mine water	−
3·2	Springs, Klein Blesbok (Ancor)	Spruit much sand	−
3·6		*B. Water with Little or No Sand Leachings*	
7·5	Krugersdorp Monument Dam	Dam rainwater, no run-in	++
7·3	Benoni Pan	Pan, rainwater, no run-in	++
7·3	Benoni, Location	Brick quarry rainwater, no run-in	++
7·1	Brakpan, Brenthurst	Stream, refuse dump and mine effluent	+
6·7	Johannesburg, Bruma	Stream sewage effluent	++
6·6	Boksburg Lake	Pond, Rand Water Board	++
6·5	Germiston, Primrose Brick Works	Dam, no run-in	+
6·5	Benoni, Northmead	Dam, storm run-in	−
6·5	Brakpan, G.G.M.A.	Compound wastes	−
6·4	Germiston, Natal Spruit near Old Barn	Stream contamination far distant	++
6·3	Johannesburg, Kose's Nursery	Reservoir sewage water	++
6·3	Randfontein N. Boundary	Spring	*Not searched*
6·3	Roodepoort, Florida Lake	Stream, run-in contamination far distant	+
6·1	Springs, Klein Blesbok	Spruit sand and pulp waste	+
6·1	Springs, Blesbok, Largo	Stream contamination far distant	−
6·1	Randfontein, Elandsfontein Drift	Spring contamination far distant	*Not searched*
6·1	Roodepoort West	Dam waste from location	+
6·0	Krugersdorp N. E. Boundary	Spring	+
6·0	Brakpan	Pan, sewage effluent	+
		C. Rain Water Tanks	
6·6	Johannesburg, 43 Forest Road	Tank near house	++
6·3	Johannesburg, 42 Field Road	Tank near house	++
6·2	Sandown	Tank near house	*Not searched*
6·1	Johannesburg, 31 Rosetta Street	Tank near house	++
5·9	Johannesburg, 40 Helvelly Road	Tank near house	++

plete control of the vector species, i.e. the reduction of the *Aedes aegypti* below 1%, could be brought about very easily with the application of modern insect vector control techniques.

Control within economical bounds of mosquito breeding on the Witwatersrand could be concentrated on the following:

(*a*) Destruction, removal or alleviation of all artificial breeding places around domestic dwellings, principally by giving attention to drains, rain water tanks, fish ponds and all surface drainage.

(*b*) Treating with larvicides on a preferential basis all water surfaces which could give rise to mosquito breeding, i.e. the carrying out of 'species sanitation.'

(*c*) Improving the present method of insect control in the industrial labour concentration areas, depending on the extensive use of residual insecticides already in use.

(*d*) The application of a further set of minor requirements, such as the stepping-up of the efficiency of the refuse collection and disposal services within the Municipal areas.

OPSOMMING

Die epidemiologie van geelkoors in Afrika is op verre na nog nie duidelik nie. Groot ontvanklike gebiede lê soms vlak naasaan ontplofbare epidemiese gebiede. Ten spyte van die groot aantal arbeiders wat heen en weer oor die Unie se grense gaan (sommige van hulle op 'n heeltemal onwettige wyse) en òf deur verklaarde geelkoorsgebiede trek òf van sodanige gebiede af kom, is geen enkele geval van die siekte tot dusver onder mense of bobbejane in die Unie aangetref nie.

Muskietopnames wat aan die Witwatersrand deur die Pesnavorsingslaboratorium gedoen is, het aangetoon dat die moontlike draers wat elders in Afrika vir die siekte geblameer word, karig hier by ons versprei is. Die voorkoms van hierdie potensiële draers word waarskynlik geaffekteer deur die sonderlinge hidrografiese kenmerke van die Witwatersrand en ook deur die heersende weerstoestande.

Met die oog op hierdie eienaardighede behoort die probleem van draer-kontrole nie veel moeilikheid op te lewer nie as die noodsaaklikheid vir sodanige kontrole ooit ontstaan.

The Secretary for Health is thanked for permission to reproduce information from Special Report No. 1/52 showing the relationship of mosquito breeding to contaminated water on the Witwatersrand.

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NOTES AND NEWS : BERIGTE

Mr. Lee McGregor, F.R.C.S., has moved his consulting rooms to 520, Lister Buildings, Jeppe Street, Johannesburg.

* * *

Mr. Sidney J. Hersch, F.R.C.S., Specialist Surgeon, has moved from Clarendon Centre to 106, Lister Building, Jeppe Street, Johannesburg. (Telephones: *Rooms*: 22-3444; *Emergency*: 22-4191).

* * *

Prof. A. S. Johnstone, of Leeds, will visit Johannesburg on 28 January 1959, when he will lecture at Medical House on *Recent Observations on the Radiology of the Gastro-Oesophageal Junction.*

* * *

Mr. C. H. Morgan, M.S.R., technical representative of the London Hospital (Ligature Department) Limited, manufacturers of surgical sutures, has recently arrived in Johannesburg on a 6 months' tour of the Union of South Africa. Mr. Morgan will visit the larger hospitals throughout the country and will look forward to meeting as many surgeons as possible with a view to discussing developments in the suture field.

Mr. Morgan's headquarters will be care of Petersen Limited, P.O. Box 5785, Johannesburg; *Telephone*: 835-7181; and at P.O. Box 38, Cape Town; P.O. Box 1684, Durban.

* * *

Dr. J. N. Jacobson, recently Professor of Radiodiagnosis at the University of Cape Town, has joined Drs. Komins, Denny, De Villiers and Berezowski in their practice of diagnostic radiology at 1 Lister Buildings, 195 Jeppe Street, Johannesburg.

Dr. J. N. Jacobson het by Drs. Komins, Denny, De Villiers en Berezowski te Listergebou 1, Jeppestraat 195, Johannesburg aangesluit in diagnostiese radiologiese praktyk. Dr. Jacobson was tot onlangs Professor van Radiologiese Diagnose aan die Universiteit van Kaapstad.

XU

4TH INTERNATIONAL CONFERENCE ON PUBLIC HEALTH EDUCATION WITH EXHIBITION

The 4th International Conference on Public Health Education will take place at Düsseldorf (German Federal Republic) from 3 to 9 May 1959. Delegates from about 60 countries representing public health education bodies will discuss and exchange their experiences. An exhibition will be held to coincide with the conference, and NOWEA Nordwestdeutsche Ausstellungs-Gesellschaft m.b.H. of Düsseldorf has been entrusted with the organization.

Both the Conference and the Exhibition will endeavour to promote the solution of health problems by the use of preventive media and methods to safeguard the health of children of school age between the ages of 6 and 18. The Congress will discuss in particular general problems of health education, the raising of health standards, leisure time activity and sport, as well as hygienic conditions, environmental improvements and mental health.

It will be of interest to all engaged in the education, training and welfare of youth, and also to physicians, social workers and public health officials, local government, ministerial bodies, associations and insurance companies, etc., whose work is concerned with these problems.

* * *

UNIVERSITY OF NATAL 1958 MEDICAL GRADUANDS

The following candidates have completed the requirements for the M.B., Ch.B. Degrees of this University:

Atcha, E. I.
Bhagwandeen, S. B.
Makgalemele, V. R.
Mdlalose, F. T.
Moreosele, A. T.
Pather, M. R. (Miss)
Piliso, T. L. (Miss)
Seedat, I. M.
Vaid, A. A. (Miss)

The following prizes have been awarded to the graduands in question:

Association of Surgeons Prize: Dr. S. B. Bhagwandeen.

Burroughs Wellcome Prize: Dr. S. B. Bhagwandeen.

Arthur Goldsmith Prize: Dr. V. R. Makgalemele.

Prize in Medicine: Drs. S. B. Bhagwandeen and I. M. Seedat.

Prize in Paediatrics: Mr. E. I. Atcha.

* * *

THE CHARLES HURWITZ SANTA CENTRE

This Centre has been named in honour of Dr. C. Hurwitz, who qualified at the University of Edinburgh in 1924 and has been actively interested in tuberculosis work for over 30 years.

He was Chairman of the Johannesburg Branch of SANTA from 1955–1958. During this period, by means of two mass miniature radiography units, a quarter of a million people were examined.

The Centre has been established on 14 acres of ground donated by the Johannesburg Municipality. The medical services will be provided by the Medical Officer of Health, Johannesburg, (Dr. J. W. Scott Millar) and his staff, including Dr. M. Goldberg, the Senior Tuberculosis Officer for Johannesburg.

Seven eighths of the cost of the building will be paid for by the Union Health Department, the remaining one eighth being the responsibility of SANTA National.

Seven eighths of the running costs will be borne by the Union Health Department, and the remaining one eighth by the Johannesburg Municipality.

* * *

A SYMPOSIUM ON THE EXTRA-CORPOREAL CIRCULATION

A symposium on the *Extra-Corporeal Circulation* will take place on Saturday, 31 January and Sunday, 1 February 1959, at the Medical School, Hospital Hill, Johannesburg, under the auspices of the Faculty of Thoracic Surgery of the S.A. College of Physicians, Surgeons and Gynaecologists.

The meetings will be open to all interested, and further particulars may be obtained from the Honorary Secretary, Mr. D. Fuller, Clarendon Centre, Parktown, Johannesburg.

Saturday, 31 *January* 1959

9.00—9.20 a.m. *Pharmacology of the Heart*: Dr. B. A. Bradlow.
9.20—9.40 a.m. *Metabolism of the Heart*: Dr. M. Zion.
10.30—11.30 a.m. *Some Problems of Pump Oxygenation*: Prof. D. Rosenberg.
11.30 a.m.—12.30 p.m. *Development of the Heart and the Great Vessels*: Prof. P. Tobias.
Development of Septal Defects: Mr. L. du Plessis.
2.15—3.00 p.m. *Demonstration of Pump Oxygenators (At the Brenthurst Clinic)*: Mr. D. N. Fuller.
3.15—4.15 p.m. *Results of Experimental Work*: Mr. C. Barnard, Mr. P. Marchand, Mr. J. C. van der Spuy.
4.30—5.15 p.m. *Open Panel Discussion.*
8.00—9.00 p.m. *Indications for Operation*: Dr. L. Braudo and Dr. L. Vogelpoel.
9.00—10.00 p.m. *Results in Clinical Cases*: Mr. W. Phillips and Mr. D. Adler.

Sunday, 1 *February* 1959

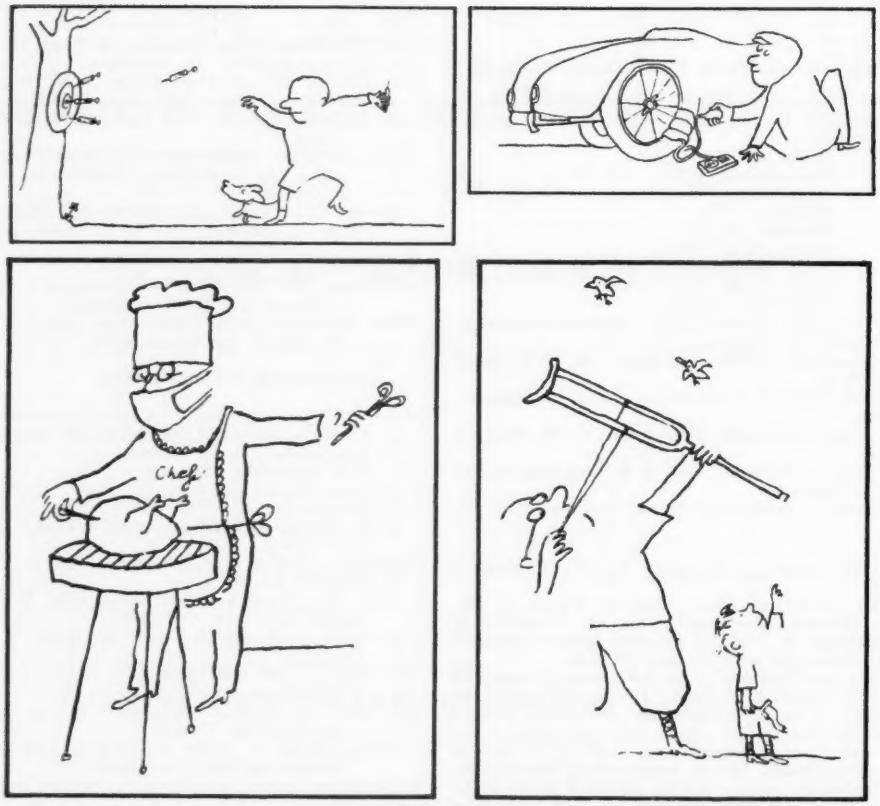
9.00—10.45 a.m. *Problems of Pump Oxygenation.*
(*a*) *Blood Collection and Storage*: Dr. M. Shapiro.
(*b*) *Anaesthesia*: Dr. B. Meaker.
(*c*) *EEG Monitoring*: Dr. Berman.
(*d*) *Blood Replacement*: Dr. W. Scott.
(*e*) *Pressures and the ECG*: Dr. M. Zion.
(*f*) *Biochemical and Haematological Control*: Dr. H. B. Greig.
11.15 a.m.—12.45 p.m. *Post-Operative Care.*
(*a*) *Surgical*: Mr. D. Adler.
(*b*) *Haematological and Biochemical*: Dr. H. B. Greig.
(*c*) *Medical*:—*Pacemaker and Monitor*: Dr. Klugman.
Cardiac: Dr. L. Vogelpoel.
Fluid Balance: Dr. W. Scott.
2.00—4.00 p.m. *Surgical Techniques*: Mr. C. Barnard and Mr. D. Fuller.
Management of Pump Oxygenators: Mr. C. Barnard and Mr. L du Plessis.

The following have been invited to preside at the various sessions:

Prof. D. J. du Plessis, Prof. J. Gear, Prof. H. W. Snyman, Mr. L. Fatti, Dr. M. Nellen, Dr. M. M. Suzman, Mr. W. H. Trubshaw, Dr. B. van Lingen, Dr. V. Wilson.

Mid-Summer Moments in the Life of the Relaxed Physician

by Tomi Ungerer



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PREPARATIONS AND APPLIANCES

NIVAQUINE

Maybaker (S.A.) (Pty.) Ltd., announce a new presentation of *Nivaquine* brand chloroquine sulphate in the form of 68 mg. (equivalent to 50 mg. base) tablets for use as a daily suppressive dose in malaria.

In West Africa where malaria is hyperendemic, experience has shown that if suppressive doses of antimalarials are not taken daily, any other dosage schedule is likely to be overlooked. The existing tablet containing the equivalent of 150 mg. chloroquine base is more than is necessary for daily suppressive dosage; moreover, there is the cost factor of the tablet of this strength and the possible increase of disagreeable side effects.

After field trials in Africa, it was decided that a tablet containing the equivalent of 50 mg. chloroquine base would be acceptable as the standard daily suppresent dose. In addition, the new 50 mg. base tablet should be useful for therapeutic use in children by overcoming the need to employ divided 150 mg. base tablets.

Besides malaria, *Nivaquine* has been used successfully in extra-intestinal amœbiasis, in lupus erythematosus, in certain vesicular dermatoses, in photosensitivity reactions such as those caused by drugs, in dermatitis, lichen planus, in the elimination of tapeworms, and in the long term therapy of rheumatoid arthritis.

SILM

Maybaker (S.A.) (Pty.) Ltd., announce the introduction of *Silm* brand fixer stain remover.

Staining is a short-coming of all liquid ultra-rapid fixers and *Silm* has been formulated so that it is suitable for eradicating photographic silver stains from all kinds of fabric, white or coloured, coarse or delicate. *Silm* is sprayed over the stained area and the marks will normally disappear within minutes of this operation. After treatment, a liberal application of water is required. Where stain is extensive, it is advisable to have the garment laundered after treatment.

Silm is likely to be of great interest to all users of photographic chemicals, for the protection and care of clothing such as white overalls.

A MOTORIZED TRACTION COUCH

Medical Distributors (Pty.) Ltd. are introducing the Rosslyn Motorized Traction Couch. This apparatus is manufactured in England by Grafton Accessories Limited and is similar in design to the well-known McManus Traction Table.

With this unit, traction may be applied to either the cervical or the lumbar spine in a rhythmic intermittent form, as sustained traction or alternatively a combination of intermittent and sustained traction. The harnesses are so well designed that they are comfortable and effective in action. The apparatus and accessories are highly adaptable and the operator may select either a prone or a supine position for the treatment. A scale to read the accurate poundage being exerted is included. The mechanism is such that the pull is applied and released gradually, allowing time for muscle relaxation and reducing the possibility of nipping peripheral nerves as they emerge from the intervertebral foramina.

Intermittent traction has been found to be of benefit in cases where there is a history of a long-standing, painful, degenerative discogenic condition. Controlled rhythmic variation, resembling a sine curve, produces alternate stretching and relaxation of muscles, ligaments and joint capsules. When the intervertebral joints separate and come together again there is an increased flow of blood and lymph due to the pumping action on these vessels.

This is the first moderately priced motorized traction table on the South African market. Several units have already been in use throughout the Union and the users have reported excellent results with all types of traction.

For Further Particulars Please Apply to the Sole S.A. Distributors:

Medical Distributors (Pty.) Ltd., P.O. Box 3378, Johannesburg and P.O. Box 3077, Cape Town.

PLEURAL BIOPSY PUNCH

Advice received from Westdene Products (Pty.) Limited is that the Abrams Pleural Biopsy Punch, manufactured by The Genito-Urinary Manufacturing Company Limited, U.S.A., is now available in South Africa.

This instrument enables a biopsy specimen to be taken from the parietal pleura, when an effusion is aspirated, without risk of damage to the lung. It is large enough to take a satisfactory specimen (4 mm. in external diameter) without causing more discomfort than ordinary aspiration under local anaesthesia. Because the biopsy is clean-cut, the histological picture is undisturbed.

The punch consists essentially of two concentric tubes and a stylet. The short trocar point is sharp enough to penetrate the chest wall after a tiny incision has been made in the skin, but it does not puncture the lung if the effusion is completely aspirated. Behind the point is a deep notch in the outer tube, which can be closed by twisting the hexagonal grip clockwise, so sliding a pin on the base of the inner tube along the oblique slot in the base of the outer tube and advancing a sharp cutting cylinder just past the notch with a rotary movement, thus cutting off and holding any tissue engaged therein. A spring clip holds the pin in either the open or closed position sufficiently firmly to prevent inadvertent movement.

Any type of syringe mount may be specified; so the punch can be used with apparatus for routine aspiration. The two tubes can be completely separated for cleaning, and as all the parts are either stainless steel or heavily plated, they can be sterilized by boiling.

Enquiries: Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

PREPARATE EN TOESTELLE

NIVAQUINE

Maybaker (S.A.) (Pty.) Ltd., kondig aan dat *Nivaquine,* 'n soort chloorchinsulfaat, nou in 'n nuwe vorm aangebied word, nl. as tablette van 68 mg. (gelykstaande aan 50 mg. van die basis), vir gebruik as 'n daaglikse onderdrukkingsdosis in gevalle van malaria.

In Wes-Afrika waar malaria hiperendemies is, het die ondervinding aangetoon dat as onderdrukkingsdosisse van die malariabestrydende middels nie daagliks geneem word nie, enige ander dosisskedule waarskynlik oor die hoof gesien sal word. Die voormalige tablet bevattende die gelyke van 150 mg. chloorchinbasis, was meer as wat 'n pasiënt vir daaglikse onderdrukkingsdoeleindes nodig gehad het; temeer, die tablette van hierdie sterkte was duur, en daar was ook die moontlikheid dat onaangename newe-effekte sou vermeerder by pasiënte wat so 'n groot dosis geneem het.

Na veldproefnemings in Afrika is daar derhalwe besluit dat 'n tablet bevattende die gelyke van 50 mg. van die chloorchinbasis aanneemlik as die standaard-onderdrukkingsdosis sou wees. Daarbenewens is die nuwe tablet met 'n basis van 50 mg. baie nuttig vir terapeutiese gebruik by kinders, want dit is nou nie langer nodig om verdeelde tablette met 'n basis van 150 mg. aan kinders te gee nie.

Afgesien van malaria is *Nivaquine* ook reeds met welslae gebruik by die behandeling van buite-ingewandsamebiase, lupus erythematosus, in sekere gevalle van blaasagtige velkwale, vir liggevoelige reaksies soos dié wat deur artsenymiddels veroorsaak word, vir huidontsteking, lichen planus, die bestryding van lintwurms, en die langtermyn-behandeling van misvormende gewrigsontsteking.

SILM

Maybaker (S.A.) (Pty.) Ltd., kondig die beskikbaarstelling aan van *Silm,* 'n preparaat vir die verwydering van die vlekke wat deur fikseermiddels veroorsaak word.

Bevlekking is 'n nadeel van al die vloeibare, ultra-vinnige fikseermiddels, en *Silm* is spesiaal geformuleer om fotosilwervlekke van alle soorte stowwe—wit of gekleur, grof of delikaat—te verwyder.

Silm word op die bevlekte oppervlakte gespuit, en, in gewone omstandighede, sal die vlekke binne tien minute na hierdie behandeling verdwyn. Hierna moet die stof in ruimskootse hoeveelhede water uitgespoel word. Waar dit 'n besonder groot vlek is, sal dit miskien raadsaam wees om die kledingstuk ná behandeling te laat was.

Silm as 'n middel vir die beskerming van klere, bv. wit oorjasse, sal groot belangstelling by alle gebruikers van fotografiese chemikalieë aanwakker.

'N GEMOTORISEERDE TREKKINGSBED

Medical Distributors (Pty.) Ltd. het die Gemotoriseerde Rosslyn-trekkingsbed beskikbaar gestel. Hierdie apparaat word in Engeland deur Grafton Accessories Limited vervaardig, en, wat ontwerp betref, is dit soortgelyk aan die bekende McManus-trekkingstafel.

Met hierdie eenheid is dit moontlik om trekking op 'n ritmiese, onderbroke manier òf op die nek- òf op die lumbale ruggraat uit te oefen—as volgehoue trekking, of, so nie, as onderbroke trekking verenig met volgehoue trekking. Die tuig is so deeglik ontwerp dat dit baie gerieflik en doeltreffend is. Die apparaat en die toebehore is besonder maklik aanpasbaar, met die gevolg dat die operateur die pasiënt òf plat op die gesig òf plat op die rug kan behandel. Daar is 'n skaal wat aandui hoeveel pond trekking uitgeoefen word. Die meganisme is so ingerig dat die trekking geleidelik toegepas en weer uitgeskakel kan word. Gevolglik is daar tyd vir die spiere om te ontspan, en die moontlikheid dat die randstandige senuwees vasgeknyp sal word namate hulle uit die tussenwerwelopenings te voorskyn kom, word verminder.

Onderbroke trekking het die bewys gelewer dat dit van waarde is in gevalle waar daar 'n voorgeskiedenis van 'n langdurige, pynlike, ontaardende, diskogeniese toestand is. Gekontroleerde ritmiese variasie wat met 'n sinuskromme ooreenstem, produseer om die beurt die uitrekking en ontspanning van spiere, pese en gewrigskapsels. Wanneer die tussenwerwelgewrigte skei en weer eens bymekaarkom is daar 'n toenemende toestroming van bloed en limf ten gevolge van die pompeffek van hierdie vate.

Dit is die eerste gemotoriseerde trekkingstafel wat teen 'n redelike prys in die Suid-Afrikaanse mark beskikbaar gestel word. Etlike eenhede word reeds dwarsdeur die Unie gebruik, en diegene wat dié eenhede op die proef gestel het, rapporteer dat hulle voortreflike resultate met alle soorte trekking behaal het.

Om nadere besonderhede doen asseblief aansoek by die Alleenverspreiders vir Suid-Afrika, Medical Distributors (Pty.) Ltd., Posbus 3378, Johannesburg, en Posbus 3077, Kaapstad.

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Hierdie instrument maak dit moontlik om 'n biopsiemonster uit die ribbeborsvlies te verkry wanneer 'n uitvloeisel opgesuig word, sonder die gevaar van beskadiging van die long. Dit is groot genoeg om 'n bevredigende monster te verkry (met 'n uitwendige middellyn van 4 mm.) sonder om meer ongerief te veroorsaak as gewone aspirasie onder 'n plaaslike narkose. Omdat die biopsie skerp omlyn is, is die histologiese beeld in geen opsig verwronge nie.

Die pons bestaan in sy wese uit twee konsentriese buise en 'n stilet. Die kort trokarpunt is skerp genoeg om deur die borswand te dring nadat 'n klein insnyding in die vel gedoen is, maar dit deursteek nie die long as die uitvloeisel volkome geaspireer word nie. Agter die punt is daar 'n diep keep in die buitenste buis wat gesluit kan word deur die seskantige klem regsom te draai. Ten

gevolge hiervan word 'n pen op die basis van die binneste buis langs die skuins gleuf in die basis van die buitenste buis geskuif, en 'n skerp snysilinder word met 'n draaibeweging tot net anderkant die keep uitgestoot, waardeur enige weefsel wat daarin is, afgesny en vasgehou word. 'n Veerknippie hou die pen stewig genoeg òf in die oop òf in die geslote posisie vas om enige onopsetlike beweging te voorkom.

Enige soort spuitmontering kan gespesifiseer word. Die pons kan gevolglik met apparaat vir roetine-aspirasie gebruik word. Dit is moontlik om die twee buise heeltemal te skei vir reinigingsdoeleindes, en aangesien al die dele òf van vlekvrye staal gemaak òf dik geplateer is, kan hulle gesteriliseer word deur hulle te kook.

Navrae: Westdene Products (Pty.) Ltd., Posbus 7710, Johannesburg.

REVIEWS OF BOOKS

HAMILTON BAILEY'S EMERGENCY SURGERY

Emergency Surgery. 7th ed. By Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.R.S.E. 1958. (Pp. 1138 + Index. With 1576 illustrations £9 9s. 0d.). Bristol: John Wright and Sons Ltd.

The name of Hamilton Bailey has long been associated with concise, comprehensive, pictorial teaching in surgery. Innumerable generations of students have found his books of outstanding clinical value in preparation for the practical side of their work.

Bailey's *Emergency Surgery* has now reached its 7th edition (38th thousand). This is eloquent testimnoy indeed to the value of this valume.

Profusely illustrated in colour, the new edition continues to place emphasis on treatment and devotes more space than before to diagnosis and differential diagnosis.

This well-indexed contribution to the practice of surgery will be a boon to every student and intern. It will undoubtedly also have a very strong appeal to the general practitioner.

HARVEY ON THE CIRCULATION OF THE BLOOD

The Circulation of the Blood. By William Harvey. The whole translated from the Latin and slightly annotated by Kenneth J. Franklin. 1958. (Pp. 185. 22s. 6d.). Oxford: Blackwell Scientific Publications.

On the occasion of the tercentenary of Harvey's death, the publishers issued a new translation of *De Motu Cordis*, prepared by Mr. Kenneth J. Franklin.

The present volume is a re-translation of *De Circulatione Sanguinis* by the same translator. The first portion of the volume is devoted to the English version of the two anatomical essays on *The Circulation of the Blood*. This is followed by several of Harvey's letters. The second half of the book is devoted to the reproduction of the original Latin text.

The principles involved in Harvey's investigation of the physiology of the circulation can well be studied with profit by contemporary students. Indeed, the intellectual adventure which Harvey undertook generates an enthusiasm and an excitement in his co-explorers even three centuries after his death.

The need to insist on a scientific approach to the problems of physiology and medicine has never diminished, and Harvey's essays are a constant reminder of the discipline to which we must unfailingly subject ourselves.

Mr. Franklin's new translation, with annotations, makes available a classic document to assist us in this objective.

JEFFCOATE'S GYNAECOLOGY

Principles of Gynaecology. By T. N. A. Jeffcoate, M.D., F.R.C.S. (Edin.), F.R.C.O.G. 1957. (Pp. 669 + Index. With 436 Figs. 12 colour plates. 84s. 6d.). London and Durban: Butterworth & Co. (Publishers) Ltd.

It is a pleasure to read a book on a clinical subject in which the author states in the preface that 'I have not played safe by stating only generally accepted views,' and then writes about his ideas which 'I do not expect all to be accepted; if they are I shall be disappointed because their object is to provoke trains of thought and discussion.'

The general lay-out of the book makes for easy reading and the happy use of headings gives the reader further evidence of the author's great ability. What a wealth of clinical experience lies behind the statement when, in discussing a mistake in diagnosis, the author writes: 'It will be found in most cases, the error lies in not attaching enough importance to what the patient told or what she could have told, had she had the opportunity.'

The clinical material and discussion are of an extremely high order and the chapter on *Genital Prolapse* is, as one would expect of Jeffcoate, really magnificent.

The book is intended for the undergraduate: nevertheless the standard is such that it can be strongly recommended to every doctor in general practice, who would do well to have a copy in his library.

By way of criticism, one would have been happier to see the author a little more enthusiastic about the use of Wertheim's hysterectomy for carcinoma of the body of the uterus. Also, having seen 5 carcinomata of the cervix in Jewesses in the last 18 months, this reviewer thinks the statement that, when this occurs, 'it has frequently been found that they have regular coitus with uncircumcised males,' is nonsensical.

CLINICAL PATHOLOGY

Clinical Pathology Data. Compiled by C. J. Dickinson, B.A., B.Sc., M.B., M.R.C.P. With a foreword by C. E. Dent, Ph.D., M.D., F.R.C.P., F.R.I.C. 2nd edition. 1957. (Pp. 81 + Index). Oxford: Blackwell Scientific Publications.

With the progressive fractionation of the practice of medicine, a concise guide to the investigations which the general physician can usefully call upon for assistance in diagnosis has become essential.

This has been provided by Dr. Dickinson in the present volume.

The inclusion of a section (by no means complete) on *Unnecessary Tests* is most timely and welcome. These are tests which 'often provide information which scarcely justifies the time and energy expended by the laboratory.' The red cell count heads this list.

The general plan is to provide, in tabular form, the normal values, and the conditions in which they are raised or lowered, followed by notes on the interpretation of the data.

This monograph stresses the value of treating the clinical pathologist as a consultant and not as a technician. If his services are integrated with the work of the clinical practitioner, the patient may often be spared expense and investigation. This will make for more efficient and accurate diagnosis. There is no longer any need for the indiscriminate application of batteries of tests whenever a new patient presents himself, a point very well brought out by the discussion on the selection of liver function tests in clinical practice (p. 63).

Tables which comprise this volume are comprehensive and systematic. They should prove of considerable value to the serious and scientific practitioner.

LECTURES ON MEDICINE

Lectures on the Scientific Basis of Medicine. Volume VI. 1956-57. 1958. (Pp. 393. With Figs. 45s.). University of London: The Athlone Press.

This book, like its annual predecessors, is composed of a series of lectures arranged annually by the British Postgraduate Medical Federation. The lectures encompass such varied subjects as *The Biological Significance of Atomic Energy, Arterial Substitutes, Cajal and Sherrington* and the *Principles of Heart Lung Machines* and can be recommended to anyone with a scientific mind and who likes to read about advances in fields other than his own. Nevertheless, some of the lectures may make heavy reading for the clinician, but if he peruses this book, his knowledge of the scientific basis of medicine will certainly be much improved.

The occasional reader will find in the *Principles of Heart Lung Machines* by D. G. Melrose a clear, succinct and an excellent review of the subject. Pumps and oxygenators are discussed on general principles by an acknowledged expert on the subject.

Arterial Substitutes by Prof. C. G. Rob gives a comprehensive account of the difficulties encountered and the encouraging advances made in the treatment of arterial narrowing with arterial substitutes.

Social Psychiatry by A. J. Lewis provides fascinating aspects of a subject which really concerns all of us, but which is sadly neglected in the medical curriculum.

Lung Function Tests by J. C. Gilson goes into a number of aspects, including recent advances, their use in surgery and also the medico-legal implications. This lecture is extremely comprehensive.

The Metabolism of Acetylcholine in Nervous Tissue by V. P. Whittaker and the *Synthesis and Degradation of Polysaccharides* by W. J. Whelan, make heavy going for the clinician, but are useful contributions to their particular subjects.

Resistance of Staphylococci to Antibiotics by Mary Barber should be read by all physicians and surgeons, and illustrates the danger of the indiscriminate use of antibiotics.

There are numerous other excellent lectures by well-known authorities and all are worth reading. Even the desultory reader who picks on one here and there will find his time well spent.

CORRESPONDENCE

NORISTAN PRIZES

To the Editor: We have pleasure in advising that Noristan Laboratories (Pty.) Limited has awarded annually, a prize of fifty guineas for the best original contribution from a general practitioner, published during any calendar year in any recognized South African medical journal. Further details are laid down in the *Rules Governing the Award of the Prize by Noristan Laboratories (Pty.) Ltd., Silverton, Pretoria.*

RULES GOVERNING THE AWARD OF THE PRIZE BY NORISTAN LABORATORIES (PTY.) LTD., SILVERTON, PRETORIA

1. A prize of fifty guineas will be awarded annually by Noristan Laboratories (Pty.) Limited for the best original contribution from a general practitioner, published during any calendar year in any recognized South African medical journal.

2. The award will be made by a Committee of experts appointed by the Board of Directors of Noristan Ltd. The decision of this Committee will be final, and no correspondence will be entered into in connexion with such decision.

3. Only original articles by general practitioners registered and practising in the Union of South Africa will be considered by the Committee.

4. The prize winner will be selected in January of each year and the award will be in respect of articles published during the previous calendar year.

5. The Committee will peruse all articles published in the journals recognized and no special application need be made by the author for any article to be considered.

6. The name of the successful medical practitioner will be published in the South African medical press.

N.B.—The prize will be awarded for the first time in January 1960.

We trust that this is of interest to you and your readers, and we should greatly appreciate it if you would publish the attached rules in your *Journal*.

H. M. Snyckers,
Director.

Noristan Laboratories (Pty.) Ltd.,
Silverton, Pretoria.

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